from a brain clot, for there would then have been some hematin or iron-bearing product. It leads to the conclusion that perhaps the hematoporphyrin in drug cases is due to metabolic (liver?) disorders, and not to a hemolysis, as has been usually supposed.

This view is supported by the fact that in hematoporphyrinuria due to drugs the liver cells show fatty degeneration.

The significance of hematoporphyrinuria apart from drug poisoning and perhaps from intestinal toxemia is not well known. It has been demonstrated by Garrod and by Soberheim[3] that hematoporphyrinuria may be present in urine after the dark color has disappeared and that, in other words, the color of the urine in this condition is not due to the hematoporphyrin, but to other abnormal pigments. It may, therefore, be that hematoporphyrinuria may be present in the urine without any change in the color. It may be inferred from the present case that its presence is not of so ominous significance as it seems to be in drug cases.

A METABOLIC STUDY OF MYOTONIA ATROPHICA.[1]

WITH THE REPORT OF TWO CASES.

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The case which forms the basis of the following considerations is interesting in several connections: (1) Because it represents a rare clinical entity; (2) because it showed in the light of metabolic study, some findings more marked than is customary in analogous conditions; (3) because one of the findings is now reported for the first time in connection with the disease; and (4) because such metabolic observations seem to throw light on the classification of the condition and suggest a means of arriving at a true diagnosis in obscure diseases of this nature.

A. B., aged thirty-seven years, white, female, married

Chief Complaint. Stiffness in the shoulder, elbow, and knee; pain in the back, and difficulty in walking

Family History. Negative, except that the patient is fairly confident that a sister who died in childbirth had an involvement of the hands similar to hers, but was otherwise healthy.

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[3] Deut. med. Woch., 1892, xviii, 566.

[1] Read at a meeting of the College of Physicians of Philadelphia, December 7, 1910.

Social History. She menstruated at the age of fourteen years, has been regular ever since, and was married at the age of twenty years. She has three children living, two of them healthy, and has had one miscarriage; one child died at birth with convulsions. A boy, aged about ten years, has hydrocephalus and is in a school for feeble-minded children. The patient states that he has some similar difficulty on opening his hands after tightly closing them, but this did not appear when he was examined. She does housework and formerly drank a small quantity of beer at intervals, but has had none for ten months; takes coffee and tea once daily.

Medical History. She has had measles and pertussis when a child, and diphtheria twelve years ago. She has never been ill in bed except for confinements and was well until the onset of the present trouble seven years ago.

Present Illness. Seven years ago she noticed a weakness in her knees which prevented her from going up stairs readily, although she otherwise felt well. With the aid of a little support she could manage easily and had no difficulty in walking on the level. The weakness in her legs became slightly more marked and one year later, after a childbirth, she developed a dull aching pain in the back. This pain has persisted on and off up to the present time. The weakness of her legs slowly increased so that four years ago she began to place her hands on her knees when she wanted to rise from a sitting posture. Three months ago she noticed some weakness in her left arm and shoulder which prevented her raising her arm over the back of her head and caused her to lean forward to comb her hair. This last development she thinks came on rather suddenly; she says she went to bed well one night and got up the next day weak, as described. She says that long before the onset of her attack she had difficulty in opening her hand after closing it tightly. When she presses her tongue against her front teeth, she cannot for a few seconds protrude it or talk. This condition she says is of recent development (a matter of a few months), though she does not know exactly how long. Her bowels have been a little loose, but she has had no difficulty in urination.

Physical Examination. This shows a rather adipose woman of large build, prominent forehead and good intelligence (Fig. 1). There is marked prominence of the seventh cervical vertebra posteriorly and the back, as a whole, is round. The tongue shows a slight moist fur and is easily and evenly protruded except as above described. The eyes give normal motions and reactions, except that the eyelids can be opened with ease against the resistance of the patient. Dentition is poor and teeth are poorly cared for.

The radial pulse is regular and of good volume, rate, and tension; the cardiac outline is normal and the sounds are good without murmur. The lungs are apparently normal. The liver extends from the fifth rib to the costal margin. The abdomen is large and relaxed, showing numerous striæ, but otherwise is negative.

Reflexes and Musculature. The eye-lids can be easily opened against the patient's resistance as mentioned, but the control of the muscles of expression is good, and there is no asymmetry of the face. No weakness of the temporals or masseter can be developed and the mouth can be drawn equally to either side, but when told to smile, the angles are not much retracted. The head is held markedly forward during the acts of sitting down or getting up. The neck is rather plump and there is a very slight swelling of the thyroid which, according to the patient, has existed in its present state for years. The sternocleidomastoids cannot be clearly felt and turning the head in various positions does not make them conspicuous. The scapulæ are both prominent on the inner edges, especially the left scapula, which protrudes markedly when the left arm and shoulder are moved. There is some evident wasting of the associated muscles, especially visible on the inner side of the scapula, but adiposity masks its exact distribution. Anteriorly the chest seems flat in the upper part and the pectorals seem less developed than

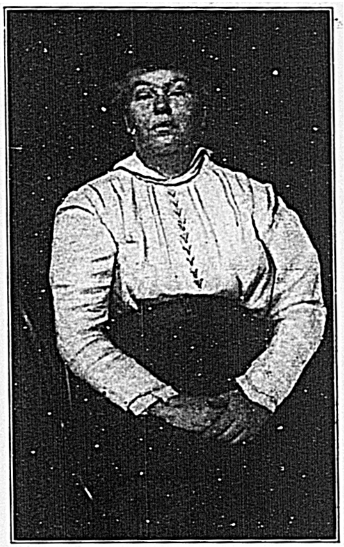

FIG. 1.—Photograph of the patient, showing the suggestion of ptosis from weakness of the levatores palpebrarum.

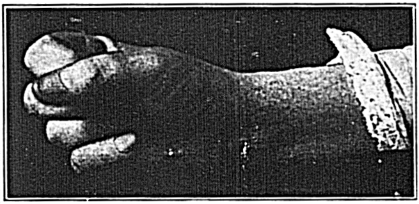

FIG. 2

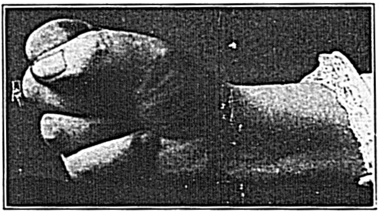

FIG. 3

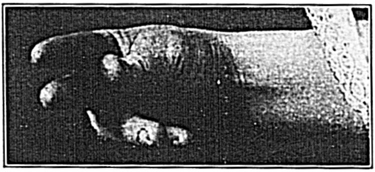

FIG. 4

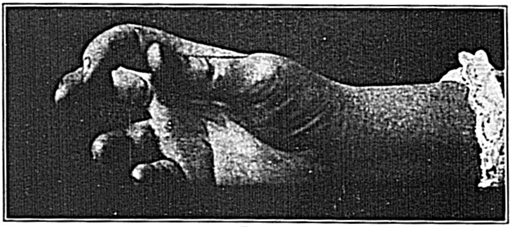

FIG. 5

FIGS. 2, 3, 4, and 5.—The pictures of the patient's hand show the successive positions assumed in the attempt to open it after forcible closure.

normally. There is no evident wasting in the deltoid, arm, or forearm region. The biceps and triceps reflexes are greatly diminished or absent. The patient has diminished but moderate strength in both arms (for acts not involving the shoulder girdle), but the shoulder muscles on the left are so weak that the left arm cannot be actively raised high enough to place her hand on her head. Passive movement of all limbs, however, is everywhere free. The hands are well formed and the fingers rather tapering. The power of contraction of both hands is below par and when closed as firmly as possible there occurs, for a few seconds, an inability to open the hands again (Figs. 2, 3, 4 and 5). When opening them, the fore and middle fingers are extended first, followed by the third and fourth in a slow athetoid manner, after which the thumb is extended and then finally the whole hand and palm are opened flat, Sometimes the third and fourth fingers are opened first, but the general movement is always the same. There is also a tendency for the thumbs to be held adducted across the hands.

The power in each leg is everywhere below normal. The left is larger than the right and has been so, she says, since the birth of her last child when she had "milk leg." It is not œdematous. The right seems to be slightly less than normal in size below the knee, there is relatively little calf, for the general build of the limb, and the muscles of the calf seem rather soft and flabby on palpation. The thighs seem smaller than they should be and there is, possibly, some flattening of the vasti externi. The left leg below the knee is, perhaps, slightly firmer to palpation than the right, but the patient feels that the power in each is about equal. The buttocks seem to be less protuberant than in health, but the gluteal crease is equal on each side and adiposity masks the true condition of the muscles here. On rising from a sitting posture, the patient places her hands on her knees to obtain greater support. In getting up from the floor she rises on one knee, then gets on all fours, finally puts out one leg and by the support of a hand on a chair, for example, rises. She cannot get up without this external aid. In walking the alignment of the feet is rather poor, though not incoördinate. The patellar jerks are diminished, though present, and there is neither Babinski's sign nor ankle clonus.

Urine. Acid, with a granular sediment, a few white blood corpuscles, and some epithelium.

On October 27 the systolic blood pressure was 96 mm. Hg. and the diastolic 65.

On October 29 the eye fundus showed no change though there was beginning opacity of both lenses.

The leukocyte count was never higher than 8650 and the differential count was normal. Hemoglobin, 88 per cent.

The patient stated (April 28, 1910) that in swallowing she sometimes has some difficulty, which is of fairly recent development.

On April 28, 1910, the electrical reactions were tested by Dr. J. W. McConnell and found to show some diminution of response, about equal everywhere, but there was no indication of the reactions of degeneration.

Weight, on October 14, 1909, 153 pounds (69.54 kilos).

Weight, on October 22, 1909, 157 pounds.

The patient is a sufferer from psoriasis which is worse in summer. On August 23, 1910, about ten weeks since last previous examination and about ten months since she first presented herself, she said she felt that she was slowly growing worse and had recently developed some unsteadiness of station which she thought was not due to weakness. She complained of some slight difficulty in swallowing. On examination, her pupillary reactions were found as before and her station with the eyes shut was good. On rising, however, or on stopping suddenly when walking, she would, at times, show the above unsteadiness. She also complained of increasing difficulty in performing her work. The thenar and hypothenar showed some suggestion of atrophy though it was not marked.

Subsequently to the completion of the metabolic studies upon the present case, here to be reported, I had the opportunity to examine another instance of the same condition. It occurred at the Philadelphia General Hospital in the service of Dr. D. J. McCarthy who has very kindly given his permission to have it included in the present paper. For this courtesy, I wish to express his appreciation.

Social and Medical History. The patient is a white male, aged forty-two years, formerly married, but only for a few months, when he left his wife for infidelity. He had no children. His family history is negative for any analogous condition, though one brother now has phthisis and one sister cannot talk loud; possibly as the result of diphtheria and some operation connected with it. The patient's birth was normal, and he was the fifth member of the family. He says that he had repeated attacks of diarrhœa until two years of age, when he developed "St. Vitus' dance." At intervals of two years afterward, and until he was twenty-six years old, he had recurrent attacks of it, for which he took Fowler's solution, up to thirteen minims three times a day, which always relieved him. Otherwise, he says, he always felt well, except that at the age of sixteen years he had "bronchitis," which confined him to bed for a month. At one time he injured his eyes on a bet by looking at the sun for five minutes at 3 o'clock of a bright afternoon. This wager resulted in blindness, which lasted three weeks, since when he has worn glasses. He says that from fourteen to nineteen years of age he was subject to headaches, which were relieved for five years by the removal of a nasal polyp, but they then recurred, and he now has a slight frontal headache much of the time. His voice is somewhat nasal. He denies all venereal history.

Present Illness. The onset of his present trouble dates back seventeen years, when he was a nurse in a large hospital. A fall called his attention to the fact that his feet flopped in walking and that his legs seemed weak. His knees soon became affected, causing him to fall more frequently and his progress from that time has been steadily worse. Ten years ago he first developed some symptoms of Thomsen's disease, seven years after the weakness in his legs began, and now experiences frequently a "locking" of the limbs when he rises from sitting or begins almost any act involving the arms or legs.

Physical Examination. Examination shows a well-nourished and intelligent man of 5 ft. 7 in. He is "pot bellied" from accentuation of the dorso-lumbar curve. The facial expression is intelligent and tranquil. The extraocular movements are apparently normal though there may be some slight weakness of convergence. The pupils are equal and react well to accommodation, but sluggishly to light. There is some ptosis when the patient looks before him. The orbiculares and levatores palpebrarum are markedly weak and there is twitching of fasciculi of the orbicularis palpebrarum on the left which, he says, has been present for seven years. There is slight weakness of the buccinators, but not of the orbicularis oris. The tongue is protruded evenly and easily in midline and is clean, without fissure, atrophy, or tremor. The sternocleidomastoids are greatly atrophied and when the head is put back it cannot be brought forward without great effort. There is free passive and active motion of both arms and coördination is good.

There is no atrophy of the subscapular group on either side, but both supraspinati show some atrophy, especially on the left. The left deltoid shows marked atrophy, but the right apparently shows none. There is slight atrophy of the biceps and triceps on the left, and a very little on the right. There is considerable loss of extensor power on the right. Flexor power of the right arm is good, of the left moderate. There is moderate atrophy of both forearms, especially of the ulnar flexor group, and the grip is reduced 50 per cent. Both thenar and hypothenar eminences show slight atrophy, and it is moderate in all the interosei. The triceps reflexes are present on both sides on reinforcement, but the biceps can be obtained only on the right.

The legs everywhere show wasting, though the right is slightly larger than the left. There is a corresponding and moderate loss of power in both legs. The knees are both hypotonic and the knee caps protrude anteriorly. The vasti externi share in the general wasting and probably suffer slightly, though not markedly, more than the rest of the leg muscles. The flexors of the foot show weakness. There is no knee jerk, ankle clonus, or Babinski sign, and there is no tenderness over the nerve trunks. The toes flex to plantar irritation.

The station with the eyes open or shut is good, though there is some instability because of weakness in the legs. There is a steppage gait. The hands show the myotonic feature of inability to relax for a few seconds after forcible closure and finally relax in a slow athetoid manner. The legs "lock," that is, become fixed in position when he starts to walk, as do the arms also for certain acts, and when the legs are affected his whole body remains apparently motionless for an instant. When the limbs are locked, he muscles concerned are contracted.

The intrathoracic and intra-abdominal organs present no important abnormalities, though both testes show atrophy, especially the left. The patient has no sexual desire and erections are involuntary. He cannot retain his urine as long as normally.

A review of the bibliography reveals the fact that but one report of this interesting condition has been made in American literature, so far as the author can ascertain,[2] although in all a total of twenty-five cases have been reported by foreign observers. In 1897, Pelizaeus called attention to a case evidencing the phenomenon of Thomsen's disease (myotonia congenita) with symptoms of muscular atrophy, and another instance was reported in 1899, by Nogues and Sirol. In 1900, Hoffman contributed a study which showed that muscular atrophy occurs in approximately 9 per cent. of cases of Thomsen's disease, since when there has appeared a number of articles recording the association of muscular atrophy with manifestations of Thomsen's disease and sometimes of myasthenia. Probably the most complete exposition is contained in a monograph by Batten and Gibb.[3] Various names have been applied to this syndrome, the present nomenclature of which is redundant and confusing to the student. Rossolimo first used the term "myotonia atrophica" because it serves shortly to describe the salient features, but this bears an unfortunate resemblance in sound to myotonia congenita (Thomsen's disease), and to myatonia congenita or amyotonia congenita. Other terms employed have been "myotonia with muscular atrophy" (Lannois), "muscular atrophy with the electrical reactions of Thomsen's disease" (Lortat, Jacob and Thaon), "Thomsen's disease with muscular atrophy" (Nogues and Sirol), "Atypical forms of Thomsen's disease" (Pelz), "Muscular atrophy with slow relaxation of muscles" (Kleist), but myotonia atrophica is that most frequently applied. The term "myotonia" is objected to by some, since it is occasionally used as synonymous with Thomsen's disease and, similarly, the use of the adjective "atrophica" has found disfavor because the order of events is not always as the title suggests, that of dystrophy followed by atrophy and also because the atrophy may not occur in the muscles affected. It is not the purpose of this article to consider at length, as has been well done elsewhere, the clinical features and variations of this disease, but the rarity of the condition demands, perhaps, a few words of exposition as to the leading characteristics.

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[2] Hunt, Ramsay. Myotonia Atrophica, Jour. Nervous and Mental Diseases, 1908, xxxv, 269.

[3] Brain, 1909, xxxii, 187.

There seems to be a familial tendency in regard to its occurrence, as might be expected in view of the hereditary nature of Thomsen's disease, but the etiology is at present quite obscure. The disease more commonly affects males between twenty and thirty, and the myotonic feature usually precedes the atrophy, though, as above indicated, this order is by no means constant. The sex of the first case here reported is interesting, since the few recorded cases leave no doubt that the disease is commoner among males. In only 3 out of 20 cases were the patients women, and in 2 of these the symptoms were so slight as to have escaped observation until it was attracted by the presence of the affection in a more marked degree in other members of the family. The condition begins insidiously, with or without a previous history of myotonic symptoms and with varying degrees of incapacity. When the case has developed, examination reveals muscular atrophy in the face, especially in the orbicularis palpebrarum and orbicularis oris, with occasional involvement of the mandibular group or the sternocleidomastoids. Of the upper extremities the forearm may be wasted, but the hand muscles are usually well preserved. The vasti of the thigh generally show atrophy, as do sometimes the peroneal and anterior tibial group, but elsewhere in the body the musculature escapes, except for a rare affection of the laryngeal. That the distribution of the atrophy may vary is well illustrated by the case of Steinert's that came to autopsy in which, contrary to the usual distribution, the hands and general musculature of the body were involved. According to Batten and Gibb, the muscles of the shoulder and upper arm are rarely affected, but in a few cases there has been noticed a wasting of the supraspinati and infraspinati. The myotonic symptoms are usually not widespread and are generally best or solely exhibited in the grasp of the hand. Flexion of the fingers and hand around an object is usually well performed, but on attempting to relax, a considerable interval must elapse before the hand can be straightened out; and even then this may be accomplished by a somewhat athetoid contortion. The greater the force of the grasp, the greater the difficulty of undoing it, though, after repeating the act a few times, it is finally easily accomplished. The difficulty of relaxation is not due to weakness of the extensors. The tongue may occasionally show involvement and there have been recorded instances in which this disability affected the upper arm and neck, but the legs are usually but slightly implicated. There are also instances in which perfectly typical cases of myotonia congenita have been affected with muscular dystrophy (Steinert), but in these the myotonia preceded the atrophy, had existed from childhood, and was widely distributed. Collins[4] has described a curious condition in which there were present extensive myotonic symptoms or purposive movements which affected the whole body and were accompanied by some hypertrophy. The myotonia, however, tended to increase as long as the movements were kept up and there was no atrophy, no myotonic reaction, or familial history. There also seemed to be some evidence of hysteria.

The electrical reactions in the disease under consideration, show a diminution of irritability to the faradic current, but nothing more. The pathology of the disease is confined to the study of one or two cases by Rossolimo and Steinert, the latter of whom has recently made a complete examination and has shown that the condition of the muscles is the same as in other myopathic cases. He found, however, degeneration of the posterior column in the lumbar region which could be traced up into the cervical, but he regards the cord condition as purely incidental. He agrees with Hoffman that these cases are true instances of Thomsen's disease and sums up his conclusions as follows: "The so-called 'myotonia atrophica" is a condition in which a typical case of Thomsen's disease is affected with muscular dystrophy. The clinical feature of this atrophy is sharply defined and highly characteristic. It occurs only in this form in Thomsen's disease. The comparative frequency of impotence and atrophy of the testicle should be emphasized." More recently a suggestive case in a female, aged thirty-four years, has been reported by Steinert in which the symptoms of the menopause had occurred. Sometimes the myotonic features are late in appearing and occasionally the myotonia has followed myopathy. Batten is inclined to place these cases in the great group of myopathies, but in his own words, further clinical and pathological experience is required before any definite statement can be made.

It will be seen from a consideration of the above that the present case can be best categoried by inclusion in the group designated myotonia atrophica, and I wish to express my obligation to Dr. William G. Spiller for helping me to a diagnosis and for emphasizing the neurological interest involved.

When the patient first presented herself at the dispensary for treatment, it seemed that observations on the general metabolism, creatinin, and calcium in particular, would be of interest and might help to classify the condition, especially in view of the results of some similar investigations conducted recently on a case of myasthenia gravis.[5]

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[4] Postgraduate, New York, 1905, xx, 516, 519.

[5] Pemberton, A Study of the Metabolism of Myasthenic Gravis, with a Suggestion Regarding Treatment, Amer. Jour. Med. Sci., 1910, cxix, 816.

In this last instance a rather low creatinin output was observed together with a very marked loss of calcium. A low creatinin output in myasthenia has been noted by Spriggs, though not so low as in the case referred to, but the calcium determinations just recorded were then made for the first time in this condition and were of considerable interest. The loss of calcium in seven days amounted to over 9 grams, and after the administration of this element for over two years by Dr. William G. Spiller, under whose care the patient was, recovery was practically complete and the patient now feels himself well. A consideration of this and of the other close relations which have been shown to exist between calcium and various muscular phenomena, emphasized the advisability of extending the observation to other muscular aberrations.

As far as I am aware no metabolic study of the calcium metabolism is on record in regard to either muscular dystrophy or Thomsen's disease, and the present case seemed a fitting one in which to attempt such a determination. Indeed, no attempt has been made to approach the question from any standpoint other than the clinical, beyond the suggestion made some years ago that veratrum poisoning in frogs caused somewhat similar phenomena on electrical stimulation, and that the condition might be referable to some metabolic disturbance. After a day or two of observation in the dispensary the patient was admitted October 7, 1910, for the purpose of diagnostic and metabolic study to the service of Dr. Joseph Sailer, at the Presbyterian Hospital, to whom appreciative acknowledgment is due for the privilege of undertaking and reporting the work upon her when in the ward.

She was placed upon a generous weighed diet, which was adapted to suit her particular demands, and analyses were made over a period of five days on the urinary nitrogen, creatinin, and calcium and the fecal nitrogen and calcium. The patient had been living under circumstances of rather poor hygiene, had been worried about herself, and had been expending much effort in the care of her home. When she was put at rest in bed in the hospital, the physical quiet and generally favorable conditions reduced the strain from which she had suffered and improved her spirits and stimulated her appetite. The results were that she gained two pounds in five days and showed a nitrogen retention of 28 grams. In this same period the calcium metabolism presented almost a balance showing a trifling loss of 0.5668 grams.

The creatinin output in the case now under consideration was found to average 0.40278 grams per diem which gives the creatinin nitrogen expressed in terms of total urinary nitrogen as 1.77 per cent.

In a series of cases of various perversions of muscular function, including dystrophy, Spriggs[6] observed an output of creatinin ranging from 0.5 gram to 3 grams, but in none recorded was it so low as 0.40278. In myasthenia gravis (my case[7]), the creatinin output was 0.39155. Sprigg's adult case of dystrophy gave a daily average of 0.53 grams which is, howevèr, a creatinin percentage as above of 1.7.

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[6] Quarterly Journal of Medicine, 1907, i, 63.

It has been found useful to observers generally to express the daily creatinin elimination in milligrams of creatinin per kilo of body weight and this ratio is called the creatinin co-efficient. In health it varies from 7 to 11 milligrams of creatinin nitrogen. In Sprigg's series he found the lowest co-efficient in the dystrophy cases which gave 2.2 and 4 milligrams respectively. The former of these was in a boy of only fifteen and three-quarter years.

Levene and Kristellar[8] have also observed very low creatinin co-efficients; and one adult, aged twenty-one years, in their series of five cases of muscular dystrophy gave a co-efficient of only 0.7, but observations were made over a period of two days only and such an extremely low result is very exceptional. The other adults in their series averaged 3.16. Similarly other observers have recorded low results in dystrophy and in a number of pathological conditions, and Myers has found a co-efficient of 2 in two very discrepit old women studied by him. This is a very low figure, however, for adults, and the case now being reported is noteworthy in that the creatinin co-efficient was 2.0139.

This was not suggested, *a priori*, by the condition or the general activity of the patient, who is able to care for her own home and a family of five. While there was plainly atrophy of tissue in the scapular region, it did not seem excessive, and was further masked by the general adiposity. There was no pseudohypertrophy. A consideration of these results was an aid in classifying the disease from which the patient suffered and indicated more strongly than could clinical observations alone, its probable nature as well as the degree of involvement. It was possible to eliminate myasthenia gravis with relative ease, although in the past myotonia atrophica has been so diagnosed; but had there been more doubt as to this, a consideration of the calcium balance would have thrown weight in the same direction. This last needs corroboration by studies of other cases, but it is cited to indicate the use to which could be put the slowly increasing number of metabolic observations in these states, and the importance of studying such doubtful affections from a dynamic point of view.

More complete observations on the creatinin and calcium metabolism in dystrophy and several muscular perversions of function are now being made.

CONCLUSIONS. As far as the study of one case can be relied upon it may be said that there is apparently no significant disturbance of the calcium output in muscular dystrophy; at least, in the presence of those phenomena which suggest Thomsen's disease. This can probably be also inferred, though not surely, in regard to Thomsen's disease "per se." The creatinin output, however, under these circumstances may be considerably reduced and the creatinin co-efficient conspicuously low. A consideration of the calcium, creatinin, and general nitrogenous metabolism as recorded here, and in myasthenia gravis, may lead to information of value in classifying doubtful conditions of this nature and localizing the perversions of function. It seems that such investigations should be more frequently undertaken in careful clinical studies.

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[7] AMER. JOUR. MED. SCI., 1910, cxl, 256.

[8] Amer. Jour. Phys., xxiv, 45.

Finally, I desire to express, with appreciation, my indebtedness to Dr. James E. Talley for many courtesies shown me in the wards of the Presbyterian Hospital.

Diet.

Oct. 13	Eggs.	Bread	Milk	Rice.	Butter.
B	88	62	12		
D	84	70	12	65.1	.55
S	85	51	12		
Oct. 14					
B	89	90	12		
D	91	70	12	57.2	99
S	89	65	12		
Oct. 15					
B	89.75	72	12		
D	85	80	12	63.90	32
S	89.50	67	12		
Oct. 16					
B	72	79	12		
D	96	72	12	69.70	89
S	84	76	12		
Oct. 17					
B	112	78.5	12		
D	97.50	80	12	51.60	60
S	109	27.5	12		
Total . .	1350.75	1040.0	180	307.50	335

Percentage Contents of Foodstuffs in Ca and N.

	Per cent. calcium oxide.	Per cent. nitrogen.
Eggs	0.122	2.008
Bread	0.041	1.265
Milk	0.184	.0.574 (1031 sp. gr.)
Rice	0.0145	1.110
Butter	0.030	0.080

Total Food Ingested.

		Grams.
Eggs.	Calcium	1.1771
	Nitrogen	27.1231
Bread	Calcium	0.3045
	Nitrogen	13.1560
Milk.	Calcium	7.8049
	Nitrogen	34.0873
Rice.	Calcium	0.0445
	Nitrogen	3.4143
Butter	Calcium	0.1073
	Nitrogen	0.2680

URINARY FIGURES FOR EACH DAY.

		Grams.
Oct. 14	590 c.c.	
	Creatinin	0.300
	Ca	0.1257
	N	6.720
Oct. 15	800 c.c.	
	Creatinin	0.386
	Ca	1907
	N	8.6240
Oct. 16	410 c.c.	
	Creatinin	0.2695
	Ca	0.1508
	N	4.6592
Oct. 17	1100 c.c.	
	Creatinin	0.5742
	Ca	0.2392
	N	12.0102
Oct. 18	1075 c.c.	
	Creatinin	0:4842
	Ca	0.2608
	N	10.2256

URINARY TOTALS.

Creatinin	2.0139
Calcium	0.9672
Nitrogen	42.2390
Ca in total feces	9.0047
N in total feces	7.5413
Total ingested calcium	9.4041
Total excreted calcium	9.9719
Negative calcium balance	0.5668
Total ingested nitrogen	78.0476
Total excreted nitrogen	49.7821
Positive nitrogen balance	28.2655
Daily average of creatinin excreted	0.40278
Total creatinin excreted	2.0139

Creatinin co-efficient = milligrams of creatinin (daily) nitrogen per kilo of body weight = (normally) 7 to 11 milligrams.

Present ratio	$\frac{0.149713}{69.54}$	0.002138

The creatinin co-efficient in the present case is, therefore, 2.138 milligrams. The figures for the creatinin co-efficient are made up on the lowest body weight; they would be even less if made up on the average body weight or on the greatest body weight attained during the experiment. The percentage contents of the foodstuffs in Ca and N was taken from "A Study of the Metabolism of Acromegaly."[9]

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[9] Edsall and Miller, Univ. Penna. Med. Bull., xvi, 143.

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CHRONIC INFLUENZA AND ITS RELATION TO NEUROPATHY.

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My thesis is that influenza may be and frequently is a chronic intoxication.

The specificity of influenza was discovered in 1902, by Pfeiffer, more than four hundred years after epidemics of the disease were first accurately described. Most authorities state that the rate of mortality is very low, and the disease has presented little of general interest aside from its pandemicity, vagaries of onset and manifestations, and its multiplicity of complications. The mode of invasion is conceded to be through the respiratory tract, and the organism is usually found in the bronchial and nasal secretions. The bacilus often persists in secretions after subsidence of symptoms in severe cases. I have been much impressed with the belief that influenza might be a chronic, as well as acute infection, or, at least, a chronic intoxication, and my beliefs, while not proved by extensive clinical experience and data, have, at least, been partially corroborated by the general history of many cases of "grippe," reported by reliable investigators. In explanation of the frequent occurrence of Bacillus influenzæ in the secretions of persons in normal health, or suffering from some other disease, such as tuberculosis, scarlet fever, measles, etc., attention may be called to the analogous occurrence of the causative organism of enteric fever, diphtheria, and cholera in persons not suffering from these diseases.